HEMILINGUAL ATROPHY OF TRAUMATIC ORIGIN.*

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Instances of hemilingual atrophy are not common, and in these the etiological element of trauma is rare. In Ascoli's[1] able summary now some ten years old, in 79 instances trauma was the cause in some 10 or 12 cases, and injury to the hypoglossal at its exit from the skull due to an injury of the cervical vertebræ was noted in about 4 instances.

The following history is reported as contributing another instance to this form of affection originating in this rare manner. I am indebted to Dr. J. Sherman Wight for the privilege of reporting it at this time.[2]

History of Patient.—A. B. W., 27 years of age (June 1, 1905), a confectioner, telegrapher, nurse, by occupation. His father, aged 62, and his mother, aged 52, were both healthy—both living and in good health. It is asserted that father had syphilis, but when, it is impossible to learn. One brother died of diphtheria; one sister is living and well at the age of 15.

Personal History.—Patient has worked since he was 12 years of age. He has had scarlet fever and measles and denies syphilis. Was a strong, healthy man. Moderate drinker. Not exposed to exogenous poisons.

History of Accident.—May 31, 1904, at 10 A. M., while a passenger on a DeKalb Ave. car on Brooklyn Bridge, he was thrown suddenly and forcibly backwards by reason of a rear collision. He was not rendered unconscious but was able to walk to a neighboring office of a patient who was about to make arrangements to hire him as a nurse. He then went home and went to bed. He called his family physician, Dr. Hoag, who said his spine was injured.

He suffered from severe pains at the end of his spine and also in the back of his head. He was shaky and tremulous. Any movement of his head resulted in severe pain. He remained in bed.

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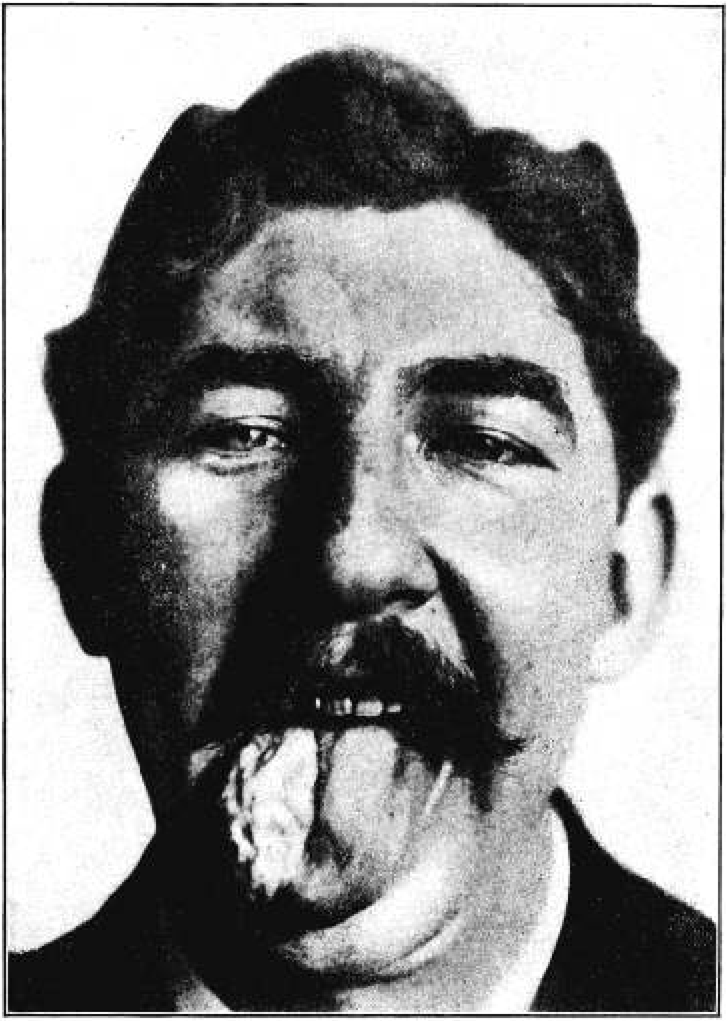
[1] Il Policlinico, I, 1894, pp. 14, 51, 159.

[2] A fuller report of this and other similar cases by Dr. Wight and myself is in preparation.

*Read by title at the meeting of the American Neurological Association, June 4 and 5, 1906.

On June 9, 1904, nine days after the accident, he was examined by Dr. Sherman Wight in consultation with Dr. Hoag. The patient was in bed. He held his head rigid with



Hemiatrophy of Tongue.

his chin forward in the position typical of cervical vertebral trauma. There was severe pain on the slightest movement and attempts at forced movement were impossible. There were no evidences of severe nerve injury at this time and patient's spine was strapped with adhesive plaster and a plaster jacket was applied. No positive diagnosis could be made at the time but absence of blood in the ear, or conjunctival ecchymoses or symptoms of involvements of cranial nerves seemed to indicate that there was no fracture of the skull. The idea was entertained that some severe injury to the cervical vertebræ was present.

The patient remained in bed seven weeks and wore the plaster jacket a period of six weeks, after which time it was removed. He stated he was unable to work, but as the accident suit against the railroad had been begun this is not unusual. He continued to complain of pain about the injured parts, held his head forward and stiff and was unable to rotate it as formerly.

From this time the history is not very distinct. The patient says that in about a month or so after the accident he noted that he did not speak as clearly as he had done previously. He thought his speech was becoming thicker, but it did not inconvenience him very much. This became worse, however, and he noted a change in his tongue, that it was unsymmetrical and quivered a great deal.

I saw him first on June 1, 1905, with Dr. Cecil MacCoy of Brooklyn. He was a moderately muscular and well built man. He carried his head forward in a stiff and strained position. Attempts at forcible movement were painful, although moderate, conscious motion was not attended with much discomfort. The only anomaly was found in the tongue. This as shown by the accompanying photograph is atrophied. It was broadened, moist, thrown up with numerous furrows, and there were very marked constant fibrillary contractions sharply limited to the atrophic side. Pinching the tongue showed a loss of muscular substance. The motion imparted by the atrophied muscles was distinctly weaker than the action of the well side.

Thus on protrusion the tongue tip was forced to the atrophied side and a typical though slight bending of the lingual raphe was observed. By strongly pulling the tongue within the mouth a certain amount of bending in the opposite direction could be induced. The movements of the tongue, however, were not strongly interfered with. A slight but distinct slurring of speech could be detected for certain labials.

There were no detectable changes in taste perception. Electrical tests were not satisfactory, as a typical reaction of degeneration was not obtained.

There were no observable changes in the innervation of the palatal muscles and none in the larynx. Slight vasomotor disturbances were noted. The patient usually had a flushed face and a slight inequality of the pupils (right dilated) was noted at my last examination. It had either been overlooked or was not present at a previous examination. The patient's general attitude was hopeful and his personality was buoyant and inclined to be boastful. His intelligence was of a medium grade only.

So far as we could ascertain no other exciting cause for the atrophy was probable. It was not a congenital affair, since it was definitely not present at the time of Dr. Wight's first examination following the accident. Tabes and syringomyelia were definitely excluded. There were no indications of either trouble. We believe that we can exclude syphilis, both from his own statement as to his non-infection, and also from the fact that as ordinarily observed the lesions incident to syphilitic involvement, whether medullary, or extra-medullary, are in the great majority of cases not so clearly confined to the hypoglossal. Isolated nuclear involvement of syphilitic origin has not yet been reported, so far as I have been able to learn, and a syphilitic exudate either meningeal, or occurring outside of the foramen, usually implicates other nerve structures and thus gives rise to a more complex syndrome.

There is ample justification for the view that a traumatic luxation of the upper cervical vertebræ may give rise to this uncomplicated picture, and the case is presented as one of simple hemilingual atrophy due to injury to the hypoglossal nerve at its exit from the skull. A Roentgen photograph shows a distinctly anomalous shadow in the region of the third cervical vertebra. It is not distinct enough, however, to be offered as conclusive evidence of the injury. In view of the cases of Uhde, Hagemann and Boettger (*Arch. f. kl. Chirurgie* 22, 1878, p. 217, Brasch, *Arch. f. Psychiatrie,* 32 1899, p. 105, Morison *Br. Med. Jl.* 1, 1888, p. 75, Parry, *Lancet* 1, 1900, p. 537, Trevelyan, *Brain* 13, 1900, p. 102), and others quoted in Ascoli's[1] list, the etiological factor is considered highly probable.